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(FILE 'HOME' ENTERED AT 19:13:13 ON 24 APR 2002)

FILE 'MEDLINE, BIOSIS, SCISEARCH, CAPLUS, PHIC' ENTERED AT 19:13:28 ON 24
APR 2002
L1 18077 S PHOSPHOROTHIOATE
L2 3060 S WR-2721 OR WR-1065 OR WR638 OR WR77913 OR WR33278 OR WR-3689
L3 196 S WR-2822 OR WR-2529 OR WR-255591 OR WR-2823 OR WR-255709 OR WR
L4 21003 S L1 OR L2 OR L3
L5 0 S 100MG/KG OR 50MG/KG OR 20MG/KG OR 30MG/KG OR 40MG/KGOR 10MG/K
L6 8425 S 100MG OR 10MG OR 20MG OR 30MG OR 40MG OR 50MG OR 60MG OR 70MG
L7 0 S K4 AND L6
L8 8 S L4 AND L6
L9 6 DUP REM L8 (2 DUPLICATES REMOVED)

=> d au ti so ab 1-6 l9

L9 ANSWER 1 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
AU Fiorentini, Giammaria (1); Giovanis, Petros; Leoni, Maurizio; De Giorgi, Ugo; Cariello, Anna; Dazzi, Claudio; Caldeo, Antonio
TI Amifostine (ethyol) as modulator of hepatic and biliary toxicity from intraarterial hepatic chemoembolization: Results of a phase I study.
SO Hepato-Gastroenterology, (March April, 2001) Vol. 48, No. 38, pp. 313-316.
print.
ISSN: 0172-6390.
AB Background/Aims: Hepatic and biliary toxicity are still significant problems after intraarterial hepatic chemoembolization for liver metastases from large bowel cancers. In about 30-60% of the patients hepatic and biliary toxicity are the limiting aspects of intraarterial hepatic chemoembolization and exclude a lot of patients from a repeated beneficial treatment. Amifostine (Ethyol) is a prodrug that must be dephosphorylated to the free thiol in which form it can detoxify free oxygen radicals generated by radiation, hypoxia and by drugs such anthracyclines, platinum analogues and alkylating agents. Amifostine as inactive prodrug is primarily metabolized at the tissue site by membrane alkaline phosphatase, which is highly active in the cell membranes of normal endothelial cells and biliary tree cells but not in the cell membranes and neovascular capillaries of tumor. When dephosphorylated to **WR-1065**, amifostine is rapidly taken up into normal liver cells by a carrier-mediated facilitated diffusion transport process. The resulting high thiol content in normal liver tissue (biliary cells and hepatocytes) compared with the negligible concentration in liver metastases from large bowel cancers probably provides for selective drug resistance to intraarterial hepatic chemoembolization protecting normal tissue and allowing full therapeutic effect on tumor. Methodology: From May 1997 we planned a phase I study in patients receiving intraarterial hepatic chemoembolization for liver metastases from large bowel cancers. We started at 200mg/m2 dissolved in 250cc of normal saline given in 15min in the intra-hepatic artery 20min before an intraarterial hepatic chemoembolization consisting of mitomycin **10mg**/m2, epirubicin-50, cisplatin-60 diluted in 10 mL of contrast media, mixed in

15 mL of lipiodol UF followed by a gelfoam powder solution until stagnation of the flow. The escalating dose, every 3 patients, was: 200 mg/m2, 250mg/m2, 300mg/m2, 350mg/m2. Results: Toxicity has been observed at 350mg/m2: 1 patient reported transient hypotension (Blood pressure 70/50mm Hg), 1 patient had skin flushing and dyspnoea. 300 mg/m2 are well tolerated and seem to reduce the level of transaminases, lactic acid dehydrogenase, and gamma-glutamyl transferase. Also the duration of necrotic damage, always observed after intraarterial hepatic chemoembolization, seems shorter compared with historical controls. Conclusions: Amifostine can be certainly administered at 300 mg/m2 as intraarterial infusion and could be a significant step to ameliorate the therapeutic ratio of intraarterial hepatic chemoembolization.

L9 ANSWER 2 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Landauer M R (Reprint); Castro C A; Benson K A; Hogan J B; Weiss J F
TI Radioprotective and locomotor responses of mice treated with nimodipine alone and in combination with **WR-151327**
SO JOURNAL OF APPLIED TOXICOLOGY, (JAN-FEB 2001) Vol. 21, No. 1, pp. 25-31. Publisher: JOHN WILEY & SONS LTD, BAFFINS LANE CHICHESTER, W SUSSEX PO19 1UD, ENGLAND.
ISSN: 0260-437X.
AB The effect of combining a radiation-protective **phosphorothioate** with another agent was investigated in an attempt to increase radioprotection and reduce toxicity, The calcium channel blocker nimodipine (NIMO) was administered alone (1 or 10 mg kg(-1)) or in combination,vith 200 mg kg(-1) of the **phosphorothioate** radioprotector **WR-151327** (WR) (S-3-(3-methylaminopropylamino)propylpho acid). Radioprotection as measured (30-day survival) of mice treated i.p. 30 min before Co-60 irradiation at a dose rate of 1 Gy min(-1)) was evaluated in CD2F1 male mice. The effects of nimodipine and **WR-151327** on locomotor activity mere investigated also in a separate group of non-irradiated mice. The LD50/30 for the Emulphor vehicle control group was 8.56. For nimodipine alone (1 or 10 mg kg-1) the LD50/30 was 8.39 and 10.21 Gy, respectively, yielding dose modification factors (DMFs) of 0.98 and 1.19, respectively. When **WR-151327** was given alone, the LD50/30 was 12.48 Gy (DMF = 1.46; P < 0.05 from vehicle). **WR-151327** combined with 1 or 10 mg kg(-1) nimodipine resulted in an LD50/30 of 12.73 GY (DMF 1.49, P < 0.05 from vehicle, and when **WR-151327** was combined with **10mg** kg(-1) nimodipine the LD50/30 was 14.29 Gy (DMF = 1,67, P < 0.001 from **WR-151327**). For either dose of nimodipine, locomotor activity did not differ from vehicle. **WR-151327** and **WR-151327** + 1 mg kg(-1) nimodipine resulted in locomotor decrements for up to 4h post-administration (P < 0.05 from vehicle), and **WR-151327** + 10 mg kg(-1) nimodipine for up to 6 h (P < 0.05 from **WR-151327**). Therefore, although there was an additive radioprotective effect when the higher dose of nimodipine was combined with **WR-151327**, the locomotor decrement was also enhanced. These results demonstrate that a combination of nimodipine and a **phosphorothioate** such as **WR-151327** may be useful as a clinical setting where behavioral and physiological side-effects can be monitored.

L9 ANSWER 3 OF 6 MEDLINE
AU Danahay H; Hill S; Natt F; Owen C E
TI The in vitro and in vivo pharmacology of antisense oligonucleotides targeted to murine Stat6.

SO INFLAMMATION RESEARCH, (2000 Dec) 49 (12) 692-9.
Journal code: B8U; 9508160. ISSN: 1023-3830.
AB OBJECTIVE AND DESIGN: This study was designed to establish whether **phosphorothioate** (PS) antisense oligonucleotides (AS-ODN) targeted to Stat6 were active in vivo in a mouse model of active sensitisation. MATERIALS: Female Balb/c mice (6-8) per group were used for in vivo study. TREATMENT: Mice were treated with active PS AS-ODNs determined in initial in vitro studies. Compounds were dosed daily (3-**30mg**/kg i.v.) over the course of sensitisation to ovalbumin. METHODS: Stat6 mRNA and protein levels were determined in the spleen after treatment (quantitative northern and western analysis respectively), in addition to serum IgE (ELISA). ANOVA was used to determine any significant differences between groups. RESULTS: Both of the AS-ODNs tested in vivo, down regulated Stat6 mRNA and protein levels in the spleen by 40-50% although there was no effect on serum IgE. These treatments also induced splenomegaly in vivo and caused splenocyte proliferation in vitro. CONCLUSIONS: The AS-ODNs used can down regulate Stat6 mRNA and protein although not sufficiently to influence IgE-levels. These effects are likely to be complicated in vivo by the immune-stimulation evident as splenomegaly.

L9 ANSWER 4 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Reyderman L; Stavchansky S (Reprint)
TI Pharmacokinetics and biodistribution of a nucleotide-based thrombin inhibitor in rats
SO PHARMACEUTICAL RESEARCH, (JUN 1998) Vol. 15, No. 6, pp. 904-910.
Publisher: PLENUM PUBL CORP, 233 SPRING ST, NEW YORK, NY 10013.
ISSN: 0724-8741.
AB Purpose, To characterize the pharmacokinetic and tissue distribution profiles of a nucleotide-based thrombin inhibitor (GS522, phosphodiester oligonucleotide, GGTTGGTGTGGTTGG) following intravenous administration to rats.

Methods. Pharmacokinetic study: 10 mg/kg, 20 mg/kg, 30 mg/kg (6 animals/dose) were administered to rats by rapid injection into the femoral vein. Blood samples were collected over a 45 minute period. Plasma concentrations of GS522 were determined using capillary gel electrophoresis with laser-induced fluorescence detection. Biodistribution Study: **10mg**/kg (400 mu l, 31.46 mu Ci/ml) of H-3-GS522 was administered to rats by rapid injection into the femoral vein. The animals were sacrificed by decapitation at 1, 5, 10, 30, 60, 360 minutes post-dose (3 rats/point), Brain, blood, duodenum, eyes, heart, kidney, liver, lungs, muscle, pancreas, skin, spleen and vein samples were collected, processed and quantitated using liquid scintillation counting.

Results. The pharmacokinetic profile declines in multiexponential manner, exhibiting extremely fast distribution and elimination (t(1/2) = 7.6-9.0 min, Cl = 22.0-28.0 ml/min, V = 83.9-132.4 ml/kg). GS522 follows linear pharmacokinetics, with the area under the curve being proportional to the dose (Rsq = 0.9744). Highest radioactivity levels were detected in kidney, liver and blood (39.7, 15.7 and 15.3% dose/ respective organ). Less than 1% of the dose was detected in the heart, spleen and lungs, and >0.3% of the dose was found in the brain and eyes. The oligonucleotide associated radioactivity was uniformly distributed between the brain regions (left and right lobe and cerebellum). Six hours following the dose

administration a statistically significant increase ($p < 0.05$) in radioactivity levels was observed in the brain, eyes, skin, liver, pancreas and vein.

Conclusions. The pharmacokinetic and biodistribution profiles of GS522 following intravenous administration to rats at three doses were characterized. The oligonucleotide associated radioactivity was widely distributed in tissues. The amount of radioactivity sharply decreased with time in most tissues. Kidney, liver and muscle were the main sites of accumulation. The oligonucleotide associated radioactivity did not cross the blood brain barrier to an appreciable extent. In addition, a statistically significant increase ($p < 0.05$) in the radioactivity levels observed in select tissues suggested a re-uptake mechanism for intact oligonucleotide or its degradation products.

L9 ANSWER 5 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
AU Maranda E (Reprint); Juszczynski P; Robak T
TI Amifostine inhibits antineoplastic activity of 2-chlorodeoxyadenosine in mice with L1210 and P388 leukemia
SO CANCER JOURNAL, (NOV-DEC 1998) Vol. 11, No. 6, pp. 309-314.
Publisher: ASSOC DEVELOPPEMENT COMMUNICATION CANCEROLOGIQUE, CANCER JOURNAL, 7 RUE GUY MOQUET, BP 8, 94801 VILLEJUIF, FRANCE.
ISSN: 0765-7846.
AB Background - The influence of 2-chlorodeoxyadenosine (2-CdA) given in combination with Amifostine on the survival time of mice with L1210 and P388 leukemia was investigated.

Methods - 192 male CD2F1 mice were used in the experiment, Mice received tumor challenges (10(6) L1210 or P388 leukemia cells) on day 0 of the experiment. All treatments were initiated on the following day as daily intraperitoneal injections. They were given 2-CdA at the doses of 20; 35 and 50 mg/kg, once a day for 5 days. Amifostine was administered at a dose of 200mg/kg, 30 minutes before the injection of 2-CdA, The drugs were given alone and in combination.

Results - The survival time of the mice bearing L1210 treated with Amifostine and 2-CdA showed survival time similar to those treated with 2-CdA alone. However, in mice treated with Amifostine and 2-CdA at the dose of **50mg**/kg, survival was significantly shorter than in animals receiving 2-CdA at the same dose. In the case of P388 leukemia, the survival time of the mice receiving Amifostine and 2-CdA at the doses of 35 and **50mg**/kg was statistically shorter than that of mice receiving 2-CdA at the same doses. Amifostine given alone did not influence the survival time of mice with L1210 or P388 leukaemia,

Conclusions - Our study revealed that Amifostine decreases the antileukemic effect of 2-CdA in murine L1210 and P388 leukemia.

L9 ANSWER 6 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Koizumi, Akio (1); Naruse, Mayumi; Hirosawa, Iwao; Ohtomo, Kazuo
TI Evidence for an acceleration of programmed cell death in bronchiolar epithelium after exposure to O,O,S-trimethyl **phosphorothioate**.
SO Journal of Occupational Health, (1996) Vol. 38, No. 4, pp. 179-185.
ISSN: 1341-0725.
AB Exfoliation of Clara cells is a prelude to pathological alterations after exposure to a variety of toxicants. The reported morphological features of exfoliating Clara cells share similarity with some types of programmed cell death (PCD). The purpose of the present study is to characterize morphological changes in Clara cells in the process of PCD in physiological and pathological conditions. We used O,O,S-trimethyl

phosphorothioate (OOS-TMP) as a lung toxicant. Morphological changes in the lungs of control rats and rats killed at 2 to 48 hr after treatment with OOS-TMP (po. **60mg**/kg) were investigated by electron microscopy. In situ DNAfragmentation was determined by 3'-OH end labeling in these rats. Immunoelectron microscopy was conducted to examine the morphological changes in Clara cells in PCD. Exfoliation of Clara cells started at 2 hr after dosing. At 6 hr, many Clara cells were sloughed. In situ DNA-fragmentation positive cells were detected in the bronchiolar epithelium of both control and treated rats. Their relative incidences increased 13 fold by 6 hr and returned to basal levels by 48 hr. In contrast, no positive cells were detected in the alveolar cells of either group. Positive cells in the bronchiolar epithelium were identified exclusively as Clara cells. The ultrastructure of the DNAfragmentation positive cells revealed similar changes in control and treated rats. DNA-fragmentation, a hallmark of PCD, was detected in Clara cells of both control and treated rats, but not in alveolar cells. The relative increases in the DNA-fragmentation positive cells suggest an acceleration in PCD after treatment with OOS-TMP. The results of this study indicate that different mechanisms of cellular death occur in Clara and alveolar cells in response to toxic insults. This difference most likely reflects the cell specific mode of the action of lung toxicants.

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=> s metastases
L10     153223 METASTASES

=> s l4 and l10
L11         61 L4 AND L10

=> dup rem l11
PROCESSING COMPLETED FOR L11
L12          27 DUP REM L11 (34 DUPLICATES REMOVED)

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     (FILE 'HOME' ENTERED AT 19:13:13 ON 24 APR 2002)

     FILE 'MEDLINE, BIOSIS, SCISEARCH, CAPLUS, PHIC' ENTERED AT 19:13:28 ON
24
     APR 2002
L1        18077 S PHOSPHOROTHIOATE
L2         3060 S WR-2721 OR WR-1065 OR WR638 OR WR77913 OR WR33278 OR WR-3689
L3          196 S WR-2822 OR WR-2529 OR WR-255591 OR WR-2823 OR WR-255709 OR
WR
L4        21003 S L1 OR L2 OR L3
L5            0 S 100MG/KG OR 50MG/KG OR 20MG/KG OR 30MG/KG OR 40MG/KGOR
10MG/K
L6         8425 S 100MG OR 10MG OR 20MG OR 30MG OR 40MG OR 50MG OR 60MG OR
70MG
L7            0 S K4 AND L6
L8            8 S L4 AND L6
L9            6 DUP REM L8 (2 DUPLICATES REMOVED)
L10      153223 S METASTASES
L11          61 S L4 AND L10
L12          27 DUP REM L11 (34 DUPLICATES REMOVED)

=> d 1-27 au ti so l12
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L12 ANSWER 1 OF 27 MEDLINE DUPLICATE 1
AU Grdina David J; Kataoka Yasushi; Murley Jeffrey S; Hunter Nancy; Weichselbaum Ralph R; Milas Luka
TI Inhibition of spontaneous **metastases** formation by amifostine.
SO INTERNATIONAL JOURNAL OF CANCER, (2002 Jan 10) 97 (2) 135-41. Journal code: 0042124. ISSN: 0020-7136.

L12 ANSWER 2 OF 27 MEDLINE
AU Ben-Josef Edgar; Han Sue; Tobi Martin; Vargas Barbara J; Stamos Beth; Kelly Laura; Biggar Sandra; Kaplan Irving
TI Intrarectal application of amifostine for the prevention of radiation-induced rectal injury.
SO SEMINARS IN RADIATION ONCOLOGY, (2002 Jan) 12 (1 Suppl 1) 81-5. Journal code: 9202882. ISSN: 1053-4296.

L12 ANSWER 3 OF 27 MEDLINE DUPLICATE 2
AU Fiorentini G; Giovanis P; Leoni M; De Giorgi U; Cariello A; Dazzi C; Caldeo A
TI Amifostine (Ethyol) as modulator of hepatic and biliary toxicity from intraarterial hepatic chemoembolization: results of a phase I study.
SO HEPATO-GASTROENTEROLOGY, (2001 Mar-Apr) 48 (38) 313-6. Journal code: GA7; 8007849. ISSN: 0172-6390.

L12 ANSWER 4 OF 27 MEDLINE DUPLICATE 3
AU Putney S D; Brown J; Cucco C; Lee R; Skorski T; Leonetti C; Geiser T; Calabretta B; Zupi G; Zon G
TI Enhanced anti-tumor effects with microencapsulated c-myc antisense oligonucleotide.
SO ANTISENSE AND NUCLEIC ACID DRUG DEVELOPMENT, (1999 Oct) 9 (5) 451-8. Journal code: CJY; 9606142. ISSN: 1087-2906.

L12 ANSWER 5 OF 27 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Hasegawa, Satoshi; Koshikawa, Naohiko; Momiyama, Nobuyoshi; Moriyama, Kayano; Ichikawa, Yasushi; Ishikawa, Takashi; Mitsuhashi, Masato; Shimada, Hiroshi; Miyazaki, Kaoru (1)
TI Matrilysin-specific antisense oligonucleotide inhibits liver metastasis of human colon cancer cells in a nude mouse model.
SO International Journal of Cancer, (June 10, 1998) Vol. 76, No. 6, pp. 812-816. ISSN: 0020-7136.

L12 ANSWER 6 OF 27 SCISEARCH COPYRIGHT 2002 ISI (R)
AU vanLaar J A M; Rustum Y M; Ackland S P; vanGroeningen C J; Peters G J (Reprint)
TI Comparison of 5-fluoro-2'-deoxyuridine with 5-fluorouracil and their role in the treatment of colorectal cancer
SO EUROPEAN JOURNAL OF CANCER, (FEB 1998) Vol. 34, No. 3, pp. 296-306. Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD, ENGLAND OX5 1GB. ISSN: 0959-8049.

L12 ANSWER 7 OF 27 MEDLINE DUPLICATE 4
AU Citro G; D'Agnano I; Leonetti C; Perini R; Bucci B; Zon G; Calabretta B; Zupi G
TI c-myc antisense oligodeoxynucleotides enhance the efficacy of cisplatin in melanoma chemotherapy in vitro and in nude mice.
SO CANCER RESEARCH, (1998 Jan 15) 58 (2) 283-9.

Journal code: CNF; 2984705R. ISSN: 0008-5472.

L12 ANSWER 8 OF 27 CAPLUS COPYRIGHT 2002 ACS
IN Zupi, Gabriella
TI Human melanoma treatments and compositions using c-myc oligonucleotides
SO PCT Int. Appl., 68 pp.
CODEN: PIXXD2

L12 ANSWER 9 OF 27 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Momiyama, N. (1); Ichikawa, Y.; Ishikawa, T.; Hasegawa, S.; Mitsuhashi, M.; Shimada, H.
TI Pharmacokinetics and biodistribution in mice of matrilysin antisense **phosphorothioate** oligodeoxynucleotide formed as a therapeutic agent for colon cancer.
SO Gastroenterology, (1997) Vol. 112, No. 4 SUPPL., pp. A617.
Meeting Info.: Digestive Disease Week and the 97th Annual Meeting of the American Gastroenterological Association Washington, D.C., USA May 11-14, 1997
ISSN: 0016-5085.

L12 ANSWER 10 OF 27 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU Mata, John E.; Joshi, Shantaram S.; Palen, Brian; Pirruccello, Samuel J.; Jackson, John D.; Elias, Nadia; Page, Todd J.; Medlin, Kristin L.; Iversen, Patrick L.
TI A hexameric **phosphorothioate** oligonucleotide telomerase inhibitor arrests growth of Burkitt's lymphoma cells in vitro and in vivo.
SO Toxicology and Applied Pharmacology, (1997) Vol. 144, No. 1, pp. 189-197.
ISSN: 0041-008X.

L12 ANSWER 11 OF 27 MEDLINE DUPLICATE 5
AU Leonetti C; D'Agnano I; Lozupone F; Valentini A; Geiser T; Zon G; Calabretta B; Citro G C; Zupi G
TI Antitumor effect of c-myc antisense **phosphorothioate** oligodeoxynucleotides on human melanoma cells in vitro and and in mice.
SO JOURNAL OF THE NATIONAL CANCER INSTITUTE, (1996 Apr 3) 88 (7) 419-29.
Journal code: J9J; 7503089. ISSN: 0027-8874.

L12 ANSWER 12 OF 27 MEDLINE DUPLICATE 6
AU Akino K; Ohtsuru A; Yano H; Ozeki S; Namba H; Nakashima M; Ito M; Matsumoto T; Yamashita S
TI Antisense inhibition of parathyroid hormone-related peptide gene expression reduces malignant pituitary tumor progression and **metastases** in the rat.
SO CANCER RESEARCH, (1996 Jan 1) 56 (1) 77-86.
Journal code: CNF; 2984705R. ISSN: 0008-5472.

L12 ANSWER 13 OF 27 MEDLINE DUPLICATE 7
AU Nip J; Rabbani S A; Shibata H R; Brodt P
TI Coordinated expression of the vitronectin receptor and the urokinase-type plasminogen activator receptor in metastatic melanoma cells.
SO JOURNAL OF CLINICAL INVESTIGATION, (1995 May) 95 (5) 2096-103.
Journal code: HS7; 7802877. ISSN: 0021-9738.

L12 ANSWER 14 OF 27 MEDLINE DUPLICATE 8
AU McDonald S; Meyerowitz C; Smudzin T; Rubin P
TI Preliminary results of a pilot study using **WR-2721** before fractionated irradiation of the head and neck to reduce salivary gland dysfunction.

SO INTERNATIONAL JOURNAL OF RADIATION ONCOLOGY, BIOLOGY, PHYSICS, (1994 Jul 1) 29 (4) 747-54.
Journal code: G97; 7603616. ISSN: 0360-3016.

L12 ANSWER 15 OF 27 MEDLINE DUPLICATE 9
AU Miele M E; Bennett C F; Miller B E; Welch D R
TI Enhanced metastatic ability of TNF-alpha-treated malignant melanoma cells is reduced by intercellular adhesion molecule-1 (ICAM-1, CD54) antisense oligonucleotides.
SO EXPERIMENTAL CELL RESEARCH, (1994 Sep) 214 (1) 231-41.
Journal code: EPB; 0373226. ISSN: 0014-4827.

L12 ANSWER 16 OF 27 MEDLINE
AU Rivoire M
TI [Cancers of the colon and the rectum: news in 1992].
Cancers du colon et du rectum: nouveautes en 1992.
SO PATHOLOGIE BIOLOGIE, (1992 Dec) 40 (9 Pt 2) 943-8.
Journal code: OSG; 0265365. ISSN: 0369-8114.

L12 ANSWER 17 OF 27 SCISEARCH COPYRIGHT 2002 ISI (R)
AU RIVOIRE M (Reprint)
TI COLORECTAL-CANCER - WHATS NEW IN 1992
SO PATHOLOGIE BIOLOGIE, (DEC 1992) Vol. 40, No. 9BIS, pp. 943-948.
ISSN: 0369-8114.

L12 ANSWER 18 OF 27 MEDLINE DUPLICATE 10
AU Kanclerz A; Chapman J D
TI Influence of misonidazole, SR-2508, RSU-1069 and **WR-2721** on spontaneous **metastases** in C57BL mice.
SO INTERNATIONAL JOURNAL OF RADIATION ONCOLOGY, BIOLOGY, PHYSICS, (1988 Feb) 14 (2) 309-16.
Journal code: G97; 7603616. ISSN: 0360-3016.

L12 ANSWER 19 OF 27 MEDLINE
AU Glover D; Glick J H; Weiler C; Fox K; Guerry D
TI **WR-2721** and high-dose cisplatin: an active combination in the treatment of metastatic melanoma.
SO JOURNAL OF CLINICAL ONCOLOGY, (1987 Apr) 5 (4) 574-8.
Journal code: JCO; 8309333. ISSN: 0732-183X.

L12 ANSWER 20 OF 27 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU MILAS L; HUNTER N; ITO H; BASIC I; TOFILON P J; PETERS L J
TI RADIOPROTECTION AND CHEMOPROTECTION AS A MEANS OF INCREASING THERAPEUTIC RATIO IN THE TREATMENT OF TUMORS AND THEIR **METASTASES**.
SO HELLMANN, K. AND S. A. ECCLES (ED.). TREATMENT OF METASTASIS: PROBLEMS AND
PROSPECTS; MEETING, LONDON, ENGLAND, OCT. 15-17, 1984. XIV+406P. TAYLOR AND FRANCIS: PHILADELPHIA, PA., USA; LONDON, ENGLAND. ILLUS. (1985 (RECD 1986)) 0 (0), 137-140.
ISBN: 0-85066-294-X.

L12 ANSWER 21 OF 27 MEDLINE DUPLICATE 11
AU Wist E A
TI Effect of the radioprotector **WR 2721** on the response of metastatic Lewis lung carcinoma colonies to alkylating agents.
SO ACTA RADIOLOGICA. ONCOLOGY, (1985 May-Jun) 24 (3) 259-62.
Journal code: 1YL; 8209606. ISSN: 0349-652X.

L12 ANSWER 22 OF 27 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU MILAS L; MCBRIDE W H; HUNTER N; ITO H

TI PROTECTION BY S-2-3 AMINOPROPYLAMINOETHYLPHOSPHOROTHIOIC-ACID AGAINST RADIATION INDUCED AND CYCLO PHOSPHAMIDE INDUCED ATTENUATION IN ANTI TUMOR RESISTANCE.
SO CANCER RES, (1984) 44 (6), 2382-2386.
CODEN: CNREA8. ISSN: 0008-5472.

L12 ANSWER 23 OF 27 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU MILAS L; HUNTER N; ITO H; PETERS L J
TI EFFECT OF TUMOR TYPE SIZE AND END POINT ON TUMOR RADIOPROTECTION BY **WR-2721** S-2-3 AMINOPROPYLAMINO ETHYL PHOSPHOROTHIOIC-ACID.
SO INT J RADIAT ONCOL BIOL PHYS, (1984) 10 (1), 41-48.
CODEN: IOBPD3. ISSN: 0360-3016.

L12 ANSWER 24 OF 27 MEDLINE DUPLICATE 12
AU Milas L; Ito H; Hunter N
TI Effect of tumor size on S-2-(3-aminopropylamino)ethylphosphorothioic acid and misonidazole alteration of tumor response to cyclophosphamide.
SO CANCER RESEARCH, (1983 Jul) 43 (7) 3050-6.
Journal code: CNF; 2984705R. ISSN: 0008-5472.

L12 ANSWER 25 OF 27 MEDLINE DUPLICATE 13
AU Milas L; Hunter N; Reid B O
TI Protective effects of **WR-2721** against radiation-induced injury of murine gut, testis, lung, and lung tumor nodules.
SO INTERNATIONAL JOURNAL OF RADIATION ONCOLOGY, BIOLOGY, PHYSICS, (1982 Mar-Apr) 8 (3-4) 535-8.
Journal code: G97; 7603616. ISSN: 0360-3016.

L12 ANSWER 26 OF 27 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AU MILAS L; HUNTER N; ITO H
TI RADIO PROTECTION AND CHEMO PROTECTION BY **WR-2721** S-2-3 AMINOPROPYLAMINOETHYL PHOSPHOROTHIOIC-ACID OF A MURINE FIBRO SARCOMA GROWING IN THE LEG OR AS LUNG MICRO **METASTASES**.
SO 24TH ANNUAL MEETING OF THE AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS, ORLANDO, FLA., USA, OCT. 25-29, 1982. INT J RADIAT ONCOL BIOL PHYS. (1982)
8 (SUPPL 1), 96-97.
CODEN: IOBPD3. ISSN: 0360-3016.

L12 ANSWER 27 OF 27 CAPLUS COPYRIGHT 2002 ACS
AU Ullrich, R. L.; Jernigan, M. C.; Yuhas, J. M.
TI Influence of **WR-2721** on metastatic tumor spread after irradiation
SO Report (1975), CONF-751001-1, 4 pp. Avail.: NTIS
From: ERDA Res. Abstr. 1976, 1(1), Abstr. No. 00686

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